PATTERNS OF OVEREATING THAT CHARACTERIZE ADDICTIVE TENDENCIES TOWARDS PALATABLE FOODS

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ABSTRACT

Previous work focusing on the notion of 'food addiction' has shown that in abundant amounts, highly-caloric- and hyper-palatable-foods can lead to addictive tendencies akin to drugs of abuse such as alcohol, nicotine, cocaine, opioids, and methamphetamine. However, such research is still in its infancy, with the notion of food addiction frequently being conflated with binge eating and obesity. The purpose of this study was to determine the unique variance accounted for in the symptom count and diagnosis of food addiction by various overeating patterns and behaviours – most importantly binge eating and grazing – after controlling for established physiological and psychological covariates. A total of 201 men and women between the ages of 20 and 50 years participated in the study. Subjects came from two cohorts: (1) York University undergraduate students, and (2) a dataset from Griffith University in Australia. Subjects of the first cohort were required to complete a self-report questionnaire booklet, and have their height and weight measured in person. Subjects of the second cohort were required to complete a self-report questionnaire online, and self-report their height and weight. A multiple regression analysis was employed using the symptom score of the Yale Food Addiction Scale. Results indicated that addictive personality traits, loss-of-control eating, reward-driven eating, and grazing each made a unique statistically significant contribution to the model. In the second stage of the analysis, a logistic regression analysis was employed using the binary diagnostic variable of the Yale Food Addiction Scale as the dependent variable. It was found that only loss-of-control eating significantly contributed to the model variance. The current findings provide novel insight into the association between a grazing pattern of overeating and food addiction, and emphasize that the Yale Food Addiction Scale symptom score and diagnosis should not be used interchangeably. Findings from this study add further support for the view that the intake of hyper-palatable foods can foster addictive-like consumption.

DEDICATION

To my grandparents,
Sima, Lev, Esther, and Michael.

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CHAPTER 1: INTRODUCTION

1.1 Rationale and Purpose

Nearly two thirds of Canadian adults are affected by overweight or obesity (Parliament of Canada, 2016; Twells, Gerogy, Reddigan, & Midodzi, 2014). In an effort to better understand this pandemic, attention has recently focused on the notion that some individuals may develop addictive tendencies towards highly palatable and calorically-dense foods (Davis, 2017). While this idea of 'food addiction' (FA) has been accepted by the general public for decades, the construct has only gained some scientific support in recent years due to convincing evidence that in abundant amounts, refined, processed, artificially-flavoured, highly-caloric and palatable foods, which proliferate in our current food environment, can lead to an addictive process akin to what occurs with drugs of abuse such as alcohol, nicotine, cocaine, opioids, and methamphetamine (Davis, 2013b; De Ridder et al., 2016; Gearhardt et al., 2011a; Koball et al., 2016; Pursey, Davis, & Burrows, 2017; Roberts, 2017). In particular, responses to these modern-day foods show remarkable similarities to responses produced by other addictive substances, in that their consumption can lead to symptoms such as loss of control (LOC), tolerance, escalation of intake, withdrawal, and/or pronounced cravings (Brunault et al., 2016; Davis & Carter; 2014; De Ridder et al., 2016; Joyner, Gearhardt, & White, 2015; Loxton & Tipman, 2017; Rogers, 2017; Valdivia, Cornejo, Reynaldo, De Francesco, & Perello, 2015).

However, unlike other addiction disorders that are recognized in the *Diagnostic and Statistical Manual* 5 (DSM-5; American Psychiatric Association, 2015), FA is yet to achieve formal diagnostic status. In an effort to operationally define FA for research purposes, Gearhardt, Corbin and Brownwell (2009a) developed the *Yale Food Addiction Scale* (YFAS) to mimic the criteria used to classify substance use and abuse in the *Diagnostic and Statistical Manual-IV*

(DSM-IV; American Psychiatric Association, 2000). Since its creation in 2009, considerable research has provided increasing validation for FA as a *bona fide* substance-abuse disorder, and has added to our understanding of risk factors for, and correlates of, addictive tendencies towards food.

Individuals who meet the diagnostic criteria for YFAS-FA exhibit higher rates of overweight and obesity (Pedram et al., 2013; Pursey, Gearhardt, & Borrows, 2016; VanderBroek-Stice, Stojek, Beach, vanDellen, & MacKillop, 2017), and are more likely to be women (Mies et al., 2017; Pursey et al., 2014). YFAS-FA individuals are also more likely to display 'addictive personality traits' such as impulsivity and negative affect (Davis et al., 2011; Koball et al., 2016; Pivaranus & Conner, 2015; VanderBroek-Stice, 2017). These individuals also show evidence of a stronger dopamine (DA) signal in the mesocorticolimbic pathways and hypersensitivity towards rewarding stimuli (Loxton & Tipman, 2017; Pedram et al., 2017; Davis et al., 2013; Davis & Carter, 2014; Pivarunas & Conner, 2015). Participants who meet the YFAS criteria are also significantly more likely than their counterparts with obesity to have binge eating disorder (BED; Davis et al., 2011; Gearhardt et al., 2012).

The considerable overlap between FA and BED is not surprising given that a key characteristic of both conditions is a LOC over the intake of certain foods – typically those that are hyper-palatable and calorically-dense (Davis et al., 2009, Davis et al., 2012; Gearhardt, Phil, White, & Potenza, 2011b; Wang et al., 2011) – and that both conditions show greater hedonic or reward-driven eating behaviours (Davis et al., 2012; Davis et al., 2013b; Pedram et al., 2017). Due to these similarities, researchers and clinicians have speculated on the nature of the relationship between FA and BED. A popular viewpoint is that among those with BED, FA may reflect a more compulsive and severe form of the disorder (Davis, 2013c; Davis, 2016a). In other

words, amongst certain vulnerable individuals, the development of binge-eating behaviours may lead to a severely compulsive and pathological condition that has strong clinical and biological parallels to a conventional addiction disorder (Davis, 2013c; Davis, 2016a). Another possibility is that untreated and/or chronic BED may develop into an addiction disorder in the same way that heavy substance use can become substance dependence over time (Davis, 2013c; Davis, 2016a).

However, an accumulating body of empirical evidence indicates that a significant proportion (approximately 50%) of those with FA do not binge eat (Davis, 2013c). Indeed, varied patterns of consumption are found in all addiction disorders (Alonso-Alonso et al., 2015). For example, while some alcoholics show patterns of regular binge drinking, others show more continual drinking throughout the day (Hoggatt et al., 2015). It is speculated that FA can likewise reflect different patterns of intake, and that relatively continual or repetitive eating throughout the day – what some have called 'grazing' – (Conceição et al., 2014; Saunders, 2004) can also be a defining feature of FA.

Grazing has been defined as the consumption of relatively small amounts of food over an extended period of time, and importantly, the inability to resist such repetitive snacking despite having intentions to stop (Carter & Jansen, 2012; Conceição et al., 2014; Saunders, 2004). Two subtypes of grazing have been proposed: (1) compulsive grazing, which exhibits LOC symptoms while snacking uncontrollably, and (2) non-compulsive grazing, which refers to repetitive bouts of mindless snack consumption. Grazing has been investigated most popularly in the context of bariatric-surgery outcomes in those with treatment-resistant obesity (Conceição et al., 2014). Here, evidence suggests that the vast majority (80%) of bariatric surgery patients experience post-operative grazing episodes (Conceição et al., 2014; Saunders, 2004).

To date, no studies have investigated 'grazing' as a possible clinical component of FA, and FA is often conflated with BED. Therefore, the purpose of this exploratory cross-sectional study was to use multiple and logistic regression analyses to estimate the unique variance accounted for in the FA symptom count and the diagnosis of FA, respectively, by various overeating patterns and behaviours including grazing, binge eating, LOC eating, and reward-driven eating, after controlling for established physiological and psychological risk factors reflected in measures of body mass index (BMI), impulsivity, and addictive personality traits. A sample of male and female adults between the ages of 20 and 50 were included in the study and recruited from the university and the larger community.

1.2 Review of the Literature

1.2.1 Evolution of the Human Food Environment

From an evolutionary perspective, energy availability was typically unreliable and subject to seasonal shortages (Davis, 2013b). Consequently, in order to facilitate survival and reproductive success during times of deprivation, humans evolved a 'thrifty genotype' and the tendency to find calorically-dense foods − particularly those high in sugar and fat content − more hedonically rewarding so as to increase the motivation to seek and consume such nutrients (King, 2015; Lieberman, 2006; Neel, 1962; Pinel, Assanand, & Lehman, 2000). While the desire to consume high-calorie foods may have been beneficial to our ancestors, this characteristic presents itself as an evolutionary 'mismatch' in today's obesogenic-food environment (Davis, 2013b; Davis, 2015). Especially when − as anthropological and sociological literature attests − eating has extended beyond the act of physiological energy acquirement, and rather, has become a reflection of cultural identities, personal philosophies, and socially-desirable interactions (Arganini, Saba, Comitato, Virgili, & Turrini, 2012; Lieberman, 2006).

The Industrial Revolution began a dramatic shift in the food environment, where novel technology allowed for artificially-flavoured, calorically-dense foods high in sugar, fat, and salt to be mass-produced (Armelagos, 2014; Gearhardt et al., 2011a). Notably, the entrance of women into the workforce radically decreased meal preparation time (Arganini et al., 2012), and increased focus on the availability of 'fast food'. In the 21st century, factors such as shift work and prolonged work hours and commutes have fostered an environment in which palatable, energy-dense food is easily and inexpensively accessible 24 hours a day, 365 days of the year (Davis, 2013b; Hebebrand et al., 2014; Jones, Conklin, Surcke, & Monsivais, 2014). Currently, McDonald's sandwiches are 90% to 119% more calorically-dense than the average serving of game meat and plant food consumed by "modern hunter-gatherers" (Smith, 2004).

It has been argued that production and consumption in today's modernized food market is guided, to some extent, by genetic proclivities, where food producers use our evolved preferences to maximize profits, and are able to manipulate consumers into buying foods that are hazardous to their well-being (Armelagos, 2014; Finlayson & Dalton, 2012; Moss, 2013; Smith, 2004). The same evolutionary propensity that caused our ancestors to seek sweet fruit high in fibre, vitamins, and minerals, or high-fat meat filled with essential fatty acids, is now causing humans to want modern-day foods filled with refined sugar and processed fats (Smith, 2004). For instance, of all packaged foods available at a large Canadian grocery retailer, about two-thirds contain some form of added sugar (Acton, Vanderlee, Hobin, & Hammond, 2017). In abundant amounts, such ingredients can contribute to diseases such as diabetes, cancer, and heart disease, and also have the potential to become addictive (Monteiro, Levy, Claro, de Castro, & Cannon, 2011; Monteiro et al., 2017).

1.2.2 The Addictive Potential of Highly Processed Foods

The evolutionary propensity to choose calorically-dense foods, mixed with the arrival of an overabundant availability of inexpensive hyper-palatable foods, has created an environment in which food consumption is no longer mainly hunger-driven (as was seen prehistorically), but instead, largely reward-driven. Compelling evidence demonstrates that the ingredients in modern-day processed and refined foods, and the concentration at which they are delivered, have the ability to mimic the effects of addictive drugs on our brain and behaviour (Davis & Carter, 2009; Gearhardt et al., 2011a; Koball et al., 2016; Roberts, 2017). Specifically, certain ingredients such as sugars and fats have been compared to drugs of abuse like alcohol, nicotine, cocaine, opioids, and methamphetamine, in that they show similarities in reward circuitry engagement in both animals and humans (Alonso-Alonso et al., 2015; Berridge, Ho, Richard, & DiFeliceantonio, 2010; Davis et al., 2011).

Similar to drugs of abuse, some ingredients are being concentrated into potent forms that offer quick bloodstream absorption, allowing them to be more hedonically rewarding (Gearhardt et al., 2011a; Schulte, Avena, & Gearhardt, 2015a). For instance, corn is now being refined into high-fructose corn syrup (HFCS), a concentrated, sweet, simple carbohydrate, that in excessive quantities is associated with insulin resistance, chronic hyperinsulinemia, and elevated blood pressure (Huang, Borensztajn, & Reddy, 2011; McChesney, 2016). Consumption of HFCS has increased exponentially in the past few decades (Barlow, McKee, Basu, & Stuckler, 2017; Bomback et al., 2010; Vos, Kimmons, Gillespie, Welsh, & Blanck, 2008), such that, of the 45,000 food products found in supermarkets, about 11,000 are enhanced by corn (Pollan, 2006). Furthermore − and similar to how nicotine is mixed with an array of other ingredients to enhance the addictive potential of cigarettes (Centers for Disease Control and Prevention US, 2010) −

food producers are combining HFCS with other addictive ingredients (Gearhardt et al., 2011c). Many processed foods contain a complex array of rewarding properties that stimulate our senses (Spence, 2012) through methods such as hydrogenating and refining, and through the use of additives such as preservatives (Armelagos, 2014). For example, in addition to HFCS, Oreo cookies contain refined flour, processed cocoa, and artificial colourings and flavourings. Portion, packaging, and tableware sizes have also increased exponentially (Marteau et al., 2015; Wansink, 2010), as seen, for example, by the 22% increase in American dinner plate sizes from 1900 to 2010, and the 250% increase from regular to jumbo-sized fast-food portion sizes (Wansink, 2010). More recent studies on food-portion trends continue to show that food servings have become "super-sized" (see Steenhuis & Poelman, 2017), and that when offered larger food servings, individuals consistently consume more food (see Hollands et al., 2015).

Some have argued that not enough evidence exists to label any food, ingredient, nutrient, or additive (with the exception of caffeine which has a corresponding "Caffeine-Related Disorders" category in the DSM-5; American Psychiatric Association, 2013), as addictive (Chao et al., 2017; Hebebrand et al., 2014; Markus, Rogers, Brouns, & Schepers, 2017). Yet, there is also considerable evidence to the contrary (see Pursey et al., 2017). Research has shown that the overconsumption of hyper-palatable foods has the ability to cause addictive-like changes such as tolerance, abuse, escalation, and withdrawal (Alonso-Alonso et al., 2015; Brunault et al., 2016; Burows et al., 2017; Gearhardt et al., 2011a). Accordingly, Spring et al. (2008) have concluded that disaccharide carbohydrates are the most addictive food substance, while Schulte et al. (2015a) consider highly-processed foods containing fat and/or refined carbohydrates to have the highest potential for developing an addictive-like consummatory behaviour, compared to other foods. As such, those with addictive tendencies towards food do not typically have a desire or

craving to ingest only one particular substance such as chocolate, but are instead drawn to highly palatable food in general (De Jong, Vandeschuren, & Adan, 2016; Hebebrand et al., 2014; Pursey, Collins, Stanwell, & Burrows, 2015; Schulte, Yokum, Potenza, & Gearhardt, 2016b).

The rewarding sensations associated with the advent of highly-palatable foods − such as reduction in stress, fatigue, and negative moods (Ahmed, Avena, Berridge, Gearhardt, & Guillem, 2013) − are now encouraging, as previously mentioned, the non-homeostatic consumption of such substances (Davis, 2011). Importantly, there is good evidence that hedonically-motivated − or as some have coined 'reward-based' (Epel et al., 2014) − eating can revoke homeostatic satiety (Alonso-Alonso et al., 2011; Begg & Woods, 2013; Kenny, 2011; Lowe & Butryn, 2007; Martin, Holsen, & Chambers, 2010). Reward-based eating has the ability to alter dopaminergic neural pathways that are associated with reward sensitivity by overriding satiety signals and subsequently creating an excessive drive to eat, causing individuals to continuously overeat (Epel et al., 2014). As such, reward-based eating has been positively correlated with BMI (Epel et al., 2014). If the difficulty to resist the hedonic urge to eat is strong enough, a feeling of LOC may emerge during a reward-based eating period (Epel et al., 2014; Lowe et al., 2016). Recurring urges may create stronger feelings of LOC, which can inadvertently increase levels of anxiety and depression, which is reminiscent of the development of a drug addiction (Lowe et al., 2016; Matherne et al., 2015; Royal, Wnuk, Warwick, Hawa, & Sockalingam, 2015; Shomaker et al., 2010; Tanofsky-Kraff et al., 2011). Therefore, it has been suggested that LOC may develop over time from constant exposure to palatable foods (Lowe et al., 2016). Likewise, evidence suggests that eating-related LOC episodes typically appear in childhood, while clinically relevant symptoms develop first in adolescence (Tanofsky-Kraff et al., 2011; Hilbert & Brauhardt, 2014).

It is important to emphasize that, as with drugs, not all foods are addictive. Foods in their natural state (e.g. fruits and vegetables) are necessary for survival and are unlikely to have any addictive potential; rather, it is the foods that have been ultra-processed to contain an abundance of refined macronutrients that possess the potential for abuse (Cariler et al., 2015; Schulte et al., 2015a; Schulte, Joyner, Potenza, Grilo, & Gearhardt, 2015b). Furthermore, not all individuals who consume a certain substance will become addicted to it (Alonso-Alonso et al., 2015; Volkow et al, 2016). For example, while 91% of Canadians consume alcohol, only about 12% exceed drinking guidelines or experience LOC consumption (Government of Canada, 2012). So, while all highly processed foods have addictive potential, individual differences determine vulnerability to the development of an addictive tendency towards food (Alonso-Alonso et al., 2015; Schulte et al., 2015b; Schulte et al., 2016b). However, since highly processed foods are so accessible and ubiquitous, they pose a substantial public health concern (Schulte et al., 2015a).

1.2.3 Food Addiction

Canada has seen a 200% increase in the prevalence of obesity since 1985, with the biggest increases occurring disproportionately in the severe classes (i.e. BMI > 40; Parliament of Canada, 2016; Twells et al., 2014). Given the lack of success of behavioural interventions such as dieting and physical exercise, in the past decade scientists have begun to focus their attention on the notion of addictive tendencies towards food to better understand this rapid rise in population weight gain (Davis, 2017; Soleymani, Daniel, Garvey, 2016; Volkow, Wang, Tomasi, & Baler, 2013).

The concept of food-related addictive behaviours has been accepted among the general public for decades, as evidenced by the presence of treatment programs dating back to the 1960s, such as *Overeaters Anonymous, Compulsive Eaters Anonymous,* and *Food Addicts Anonymous*

(Davis, 2013b; Davis & Carter, 2014). The term, however, only began to gain scientific credibility in the 21st century, due to convincing scientific evidence and concurrent media press that the hyper-palatable foods in our current food environment have addictive characteristics akin to drugs of abuse (Avena, Rada, & Hoebel, 2008; Davis, 2013b; Davis & Carter; 2014; Gearhardt et al., 2011a; Hebebrand et al., 2014; Gearhardt et al., 2011a; Meule, de Zwaan, & Müller, 2017; Rogers, 2017; VanderBroek-Stice, 2017). Of most importance was the creation of the YFAS, which was developed in 2009 as a useful standardized tool for identifying FA tendencies (see Gearhardt et al., 2009), and has since fostered a noticeable increase in FA research (Davis, 2017; Pursey et al., 2014). The YFAS is based closely on the criteria used to classify substance dependence in the DSM-IV of the American Psychiatric Association (Gearhardt et al., 2009). Since its creation, the YFAS has been validated as a strong tool in identifying food-related addictive tendencies, further reinforcing that FA is a legitimate construct with pathognomonic and dopaminergic symptoms similar to drug-addiction disorders (Davis et al., 2011; Gearhardt et al., 2011a; Gearhardt et al., 2011c; Pursey, Collins, Stanwell, & Burrows, 2016).

In a population study conducted in Newfoundland, the prevalence of YFAS-FA was estimated to be 7% in females and 3% in males (among a sample of 652 adults; Pedram et al., 2013). It has also been found that YFAS diagnoses are significantly higher in those with overweight and obesity (Gearhardt et al., 2012; Eichen, Lent, Goldbacher, & Foster; 2013; Long et al., 2015; Pursey, Gearhardt, & Borrows, 2016; VanderBroek-Stice, 2017), compared to normal- or under-weight individuals (Eichen et al., 2013; Pedram et al., 2013) and to the general population (Brunault et el., 2016; Long, Blundell, & Finlayson, 2015). In addition, participants who meet YFAS criteria are also significantly more likely to meet BED criteria (reported at

about 50%; Davis et al., 2011; Gearhardt et al., 2012) and to have type 2 diabetes (Raymond & Lovell, 2015). Those with YFAS-FA also show significantly greater rates of binge eating, hedonic eating, snacking, and food cravings (Gearhardt et al., 2011c; Davis et al., 2011; Long et al., 2015).

There is still not consensus on the validity of using FA as the appropriate diagnostic terminology to describe the consumption of hyper-palatable foods in an addictive-like manner (Chao et al., 2017; Markus et al., 2017; Nolan, 2017; Nolan, 2017; Schulte, Potenza & Gearhardt, 2017). Traditionally, the term 'addiction' was used to describe the manifestation of a neurobiological substance dependency (Hone-Blanchet & Fecteau, 2014). Only recently has the term been extended to also describe behavioural manifestations such as gambling, shopping, and internet use (Hone-Blanchet & Fecteau, 2014; Davis & Carter, 2009), although according to the DSM-5 only gambling is included in this category (American Psychiatric Association, 2013). Of recent concern is the question of whether to continue using FA to describe a 'substance dependence', or to alternatively follow the behavioural model of addiction, reflecting an eating-, rather than food-based behaviour (Hebebrand et al., 2014; Markus et al., 2017; Nolan, 2017; Schulte et al., 2017). One of the biggest arguments against the substance-dependence model is that unlike other drugs of abuse, food is needed for human survival (Long et al., 2015). However, proponents of the current term view this is as a flawed perspective (Davis, 2014; Schulte et al., 2015b; Schulte et al., 2017). As mentioned before, while food is indeed essential for our existence, it is the foods that come from nature (e.g. fruits, vegetables, fish) that are needed for survival, rather than the highly-processed foods (e.g. hot dogs, pizza, donuts) which are referred to in the FA paradigm (Davis, 2014; Schulte et al., 2015a). Additionally, current evidence pointing towards the idea that the act of eating itself can be addictive is weak, while evidence for

the notion that certain foods can posit addictive-like consumption is stronger (Schulte et al., 2017). Both pre-clinical and clinical research show that today's foods are inherently addictive and have the ability to alter neural activity, particularly with regards to DA in the brain reward pathways, showing strong biological and behavioural similarities to substance abuse (Davis & Carter, 2009; Rogers, 2017).

1.2.3.1 Pre-clinical Research

The observation that addictive drugs alter the expression of DA in neurochemical reward pathways is well-established (Nutt, Lingford-Hughes, Erritzoe, & Stokes; Volkow, 2012; Volkow, Koob, & McLellan, 2016; Volkow & Morales, 2015). Animal studies have also demonstrated that the chronic consumption of a variety of foods such as sugar and corn oil mimic the altered pathways seen in drug-dependent animals (Avena & Hoebel, 2003; Carlin et al., 2016; Carter et al., 2016; DiFeliceantonio, Mabrouk, Kennedy, & Berridge, 2012; Furlong, Jayaweera, Balleine, & Corbit, 2014; Valdivia et al., 2015). As with addictive drugs, over-consuming palatable food has the ability to produce excessive extracellular DA levels in the nucleus accumbens (NAc; Carlin et al., 2016; Michopoulos, Diaz, & Wilson, 2016; Preedy, 2016) and the mesocorticolimbic network, and can lead to the same neuroadaptations seen in drug abuse (Davis & Carter, 2009). Significant changes in DA expression and increased food consumption have been noted in rats both on high fat and high sugar diets (Avena & Hoebel, 2003; Carlin et al., 2016; DiFeliceantonio et al., 2012; Vucetic, Carlin, Totoki, & Reyes, 2012). In a particularly noteworthy study, elevations in reward thresholds and reduced striatal dopamine receptor density were found in rats with extended access to an energy-dense 'cafeteria' diet consisting of foods such as bacon, sausage, cheesecake, pound cake, frosting, and chocolate (Johnson & Kenny, 2010). More recently, Soto et al. (2015) studied the effects of sucrose-

sweetened water (SSW) in rats on a high- or normal-fat diet. Interestingly, the authors found that while rats on both diets preferred SSW to unflavored water, rats on the high-fat diet showed a higher preference for the SSW and increased both their food and SSW intake over the 5-week experiment, reinforcing the notion that access to a hyper-palatable diet results in excessive food intake.

Several lines of evidence show that LOC, withdrawal, cravings, tolerance, and relapse – all of which are defining characteristics of a drug addiction – are also characteristics of FA (Avena & Hoebel, 2003; Iemolo et al., 2012; Martire et al., 2014). In the face of excessive drug use, tolerance, escalation, or habit formation occurs, meaning more of the drug is needed to produce the same effect (Volkow, Fowler, & Wang, 2002). This phenomenon has been shown in both sugar-fed and fat-fed rats (Avena & Hoebel, 2003; Valdivia et al., 2015). For example, Valdivia et al. (2015) found that rats with time-limited access to a high-fat diet tend to display escalating hyperphagia. Studies also show that a drug-like downregulation occurs when palatable food is over-consumed; these neuroadaptations also induce drug sensitization, cravings, and contribute to withdrawal (Avena & Hoebel, 2003; Epstein & Shaham, 2010; Grigson, 2002; Johnson & Kenny, 2010). In particular, opiate-like withdrawal symptoms such as teeth chattering, forepaw tremor, head shaking, anxiety, depression, and stress have been evidenced in rats upon sugar- (Iemolo et al., 2012; Martire et al., 2014) and fat-diet (Satta et al., 2016) termination. One hallmark of addiction is the inability to halt substance consumption despite experiencing negative consequences such as social, psychological, or physical problems (Epstein & Shaham, 2010). Studies have also shown that rats with extended access to a cafeteria-like diet (Johnson & Kenny, 2010) or fat (Avena, 2010) will tolerate a foot shock and will not reduce food intake.

Importantly, in a study where drug- and sugar-naïve rats were given the option between saccharin sweetened water and cocaine, 97% showed a preference for saccharin, finding that the sweetened water may be more addictive and rewarding than cocaine (Lenoir, Serre, Cantin, & Ahmed, 2007). A more recent study also reinforced the notion that sugar can be more addictive and rewarding than cocaine or heroin (Madsen & Ahmed, 2014). Using Pavlovian conditioning, the study found that rats approached and pressed a lever that dispensed saccharin sweetened water much more often than they did a lever that delivered cocaine or heroin, concluding that sugar sweetened water is more rewarding and thus more addictive than the other two drugs. Many have argued that sugar meets the criteria of any addictive substance, and can give rise to compulsive behaviours when over consumed (Avena et al., 2008; Carter, 2016; Davis, Levitan, Kaplan, Kennedy, & Carter, 2014; De Jong, Vandershcuren, & Adan, 2016; Moss, 2013).

1.2.3.2 Clinical Research

In certain individuals, overeating can display clinical similarities to addiction disorders, notably in the role of DA function. The DA pathway is responsible for reward regulation and inhibitory control, systems which play a vital role in the development of a substance addiction (Koob & Volkow, 2016; Volkow, 2012; Volkow et al., 2016). When consuming an addictive substance, acute levels of extracellular DA released in the NAc results in sensitization of the dopaminergic pathway, subsequently increasing drug salience and the desire to consume said drug (Koob & Volkow, 2016; Volkow, 2012; Volkow et al., 2016). Repeated drug exposure downregulates striatal DA receptors, creating an anhedonic state in which more of the drug is needed to produce the same levels of reward, causing hypersensitivity towards the rewarding stimuli, or what is clinically referred to as tolerance or escalation (Blum et al., 2000; Koob & Volkow, 2016; Volkow, 2012; Volkow et al., 2016). Biological and behavioural similarities

regarding DA dysfunction have been established between individuals with YFAS-FA tendencies and other substance disorders, as seen by the following research.

Similar to other addiction disorders, individuals with FA symptoms tend to display hypersensitivity towards rewarding stimuli (Davis & Carter, 2014; Davis et al., 2013; Gearhardt, Davis, Kuschner, & Brownell, 2011a; Volkow et al., 2013). For example, Gearhardt et al. (2009) found that the neural profiles of individuals with FA are comparable to those seen in individuals with addictions to alcohol, nicotine, and heroin. Both groups of individuals showed enhanced activation of the DA reward pathway (dorsolateral prefrontal cortex, anterior cingulate cortex and amygdala), while showing reduced signaling in regions responsible for inhibiting rewarding stimuli (medial orbitofrontal cortex; Gearhardt et al., 2009). Loxton and Tipman (2017) also found that YFAS-FA scores were associated with elevated reward sensitivity. Likewise, a study comparing FA to alcohol addiction found that those with high YFAS scores showed patterns of "addiction brain activity" similar to those with alcohol addiction, involving pathological responses in the dorsomedial prefrontal cortex and the medial orbitofrontal cortex (de Ridder et al., 2016).

As with other addiction disorders, genetic predispositions in the form of DA polymorphisms – both single and pooled – have also been associated with FA (Davis et al., 2013; Pedram, Zhai, Gulliver, Zhang, & Sun, 2017). While one genome-wide investigation study found no common genes or single nucleotide polymorphism candidates between FA and other drug addictions (Cornelis et al., 2016), a more recent study identified two genes linked to FA development, both of which have also been previously implicated with the dopaminergic pathway (Pedram et al., 2017). Using exome sequencing, the two genes identified included the DRD2 dopamine receptor gene, associated with an increased risk for substance use and

dependence, and the TIRAP gene, which has been shown to over-express after alcohol, nicotine, and caffeine exposure in mice (Pedram et al., 2017). In a study by Davis et al. (2013), individuals with a YFAS-FA diagnosis had genetic profiles indicative of enhanced DA signaling compared to their age- and weight-matched controls (Davis et al., 2013). A higher score was also related to higher levels of emotional overeating and food cravings (Davis et al., 2013).

Similar to those with other addictive disorders, YFAS-FA individuals also experience other addiction-related symptoms such as: (a) continued consumption despite aversive consequences and despite a desire to halt consumption (Brunault et al., 2016; Gearhardt, Corbin, & Brownell, 2009b; Meule, Heckel, & Kübler, 2012); (b) psychological distress (Afton et al., 2016; Brunault et al., 2016; Burows et al., 2017); (c) LOC (Gearhardt et al., 2009; Meule, Heckel, & Kübler, 2012); (d) withdrawal and high rates of relapse (Brunault et al., 2016; Davis, 2013b); and (e) strong cravings (Davis et al., 2013; Joyner et al., 2015). The definition of 'craving' originally derived from drug-addiction research, but the construct is now being applied to food as well, where a 'food craving' is defined as an intense desire to consume a particular food (Cepeda-Benito, Gleaves, Williams, & Erath, 2000; Rodríguez-Martín & Meule, 2015; White et al., 2002). Cravings for drugs of abuse such as tobacco, cocaine, and heroin have been shown to predict increased compulsive use (Epstein & Shaham, 2010). Similarly, the presence of food cravings has been associated with increased food consumption (Gearhardt et al., 2011a; Joyner et al., 2015) and higher food-related addictive behaviours (Polk, Schulte, Furman, & Gearhardt, 2017). YFAS-FA participants reported stronger cravings after tasting their favourite snack, compared to their non-addicted counterparts (Davis et al., 2013). Using functional magnetic resonance imaging (fMRI), Gearhardt et al. (2011) also found that in response to anticipated palatable-food intake (chocolate milkshake), those with higher YFAS-FA symptom

scores displayed greater activation in brain regions associated with reward including the left anterior cingulate cortex, orbitofrontal cortex, and amygdala.

While it is true that altered and enhanced neural reward pathways, particularly in DA signaling, play a major role in the individual vulnerability of developing an addiction disorder, the predicted outcome does not always occur (Davis et al., 2012; Davis et al., 2013; Volkow, 2012; Volkow et al., 2016). That is, having an increased neurological or genetic risk of developing an addiction disorder does not always predict its phenotypic characteristics (Volkow, 2012). Rather, it is the confluence of factors including genetics, environmental exposures, mental illnesses and personality traits that contribute to addiction susceptibility (Volkow, 2012; Volkow et al., 2016).

1.2.4 Personality Risk Factors

With the etiology of addiction being so heterogeneous among individuals, attempts to identify an 'addictive personality' have been met with much debate (Amodeo, 2015; Eysenck, 1997; Lester & Narkuski, 1978), especially in the absence of any distinctive biomarkers for the condition (Clark, 2015; Davis, 2016b). In other words, does there exist a certain set of predisposed personality 'traits' that makes one more susceptible to developing an addiction, or, are certain traits simply a consequence of the addiction? As Amodeo (2015) has argued, addictive personality traits arise from, rather than predict addictive behaviour. Some support can be attributed to this statement, since it is known that addictive substances have the potential to alter neural activity and subsequently increase the addictive behaviour, via an increase in higher compulsiveness (Volkow et al., 2016). As such, Szalavitz (2015) has argued that the entire concept of an addictive personality is a "myth", claiming that it is the interplay of a variety of genetic and environmental factors that may − or may not – cause someone to develop an

addiction disorder; or, that there is no set of personality traits that can conclusively predict addiction development. For example, antisocial personality disorder is a heritable trait that has been linked to addiction disorders, yet most individuals with antisocial personality disorder do not develop an addiction (Szalavitz, 2015).

However, about 50% of substance-use disorders are linked to a psychiatric diagnosis, sometimes in the form of a personality disorder (Szalavitz, 2015), and, researchers have managed to isolate certain personality characteristics that are more likely to predict a broad range of addictive behaviours (Bellin et al., 2016; Davis et al., 2017; Ipser et al., 2015; Meule et al., 2017; VanderBroek-Stice, 2017). That is not to say that the personality characteristics attributed to addiction are unique predictors. Indeed, it is clear that some of these traits are associated with a broad spectrum of psychopathology (Szalavitz, 2015). For instance, the trait of impulsivity has not only been linked to addictive behaviours, but also to 18 other diagnoses in the DSM-5 (e.g. ADHD, bulimia, BED, borderline personality disorder; Sperry, Lynam, Walsh, Horton, & Kwapil, 2016).

Impulsivity is a multifaceted construct (Sperry et al., 2016), primarily defined by the tendency to engage in inappropriately risky actions (de Wit, 2009; Potenza & de Wit, 2010). It has been associated both with a strong drive for reward, and an inability to delay gratification (Davis, 2017). For example, an individual with ADHD may display impulsivity through frequent interruptions and an inability to wait for their turn (Sperry et al., 2016), while an individual with FA may display a strong urge for highly palatable foods (Davis, 2013b; Murphy et al., 2014).

Several studies have found a positive correlation between impulsivity and YFAS score, where YFAS-diagnosed women showed a faster response to food cues than to neutral cues (Gearhardt et al., 2011c; Meule et al., 2012), and, where YFAS-diagnosed versus control

participants showed attentional hypervigilance towards food images (Frayn, Sears, & von Ronson, 2016). Davis et al. (2011) found that in a sample of individuals with obesity, those with co-morbid FA were more impulsive. Other studies have also found that higher levels of impulsivity predicted greater FA symptom endorsement (Davis et al., 2011; Murphy, Stojek, & MacKillop, 2014). More specifically, dimensions of impulsivity found in those with YFAS-FA included attentional impulsivity (Meule et al., 2012; Meule et al., 2017; Raymond & Lovell, 2015), motor- and non-planning impulsivity (Raymond & Lovell, 2015), negative urgency, lack of perseverance (Murphy, Stojek, & MacKillop, 2014; Pivarunas & Conner, 2015), and lack of premeditation (Murphy et al., 2014). Interestingly, in a sample of eating disorder patients, Wolz et al. (2016) found that those with co-morbid FA showed a distinct personality profile, in that they displayed lower levels of perseverance and acted more rashly in response to negative affect (i.e. negative urgency). The different facets of impulsivity displayed in these studies highlights its complexity.

Anxiousness is another multidimensional personality trait that has been correlated in both the development and withdrawal of substance (Homberg et al., 2013; Ipser et el., 2015) and behavioural (Hodgins et al., 2012) addictions (see Davis 2016b). A common viewpoint is that drug-intake is used as a coping mechanism to mitigate symptoms associated with anxiety and distress (see Bellin et al., 2016; Khantzian, 2013). Conversely, anxiousness may increase addiction withdrawal severity and promote relapse (Khantzian, 2013). YFAS diagnosed FA patients are more likely to endorse the trait of anxiousness (Koball et al., 2016). Likewise, compared to BED controls, patients with co-morbid YFAS-FA were more likely to display negative affect (Gearhardt et al., 2012; Gearhardt, White , Masheb, & Grilo, 2013), an emotional state associated with both anxiety and withdrawal (Davis, 2017; Koob & Volkow, 2016).

The notion of the 'addictive personality' has generated the need to create a clinically relevant way of identifying whether this predisposition indeed exists. In turn, various psychometric self-report scales have been developed by comparing those with drug addictions to controls. For example, the *Addiction Scale* of the *Eysenck Personality Questionnaire* (EPQ; Eysenck & Eysenck, 1975) – which was empirically derived by its predecessors including the Maudsley Personality Inventory (Eysenck, 1959) and the Eysenck Personality Inventory (Eysenck & Eysenck, 1964a) – was developed by comparing those with drug addictions to controls on traits of impulsivity, negative affect, emotional reactivity, and anxiousness (Gossop & Eysenck, 1980). The scale has since been validated in numerous countries (see Aluja, García, & García, 2003) and in various addictive behaviours such as alcoholism (Ogden, Dundas, & Bhat, 1988), gambling (Clarke, 2003), BED (Davis & Carter, 2009), and FA (Davis et al., 2011).

1.2.5 Links Between Food Addiction and Binge Eating

The strong overlap between FA and BED raises the question of whether there are any clinically relevant differences between the two conditions. Originally, binge eating was classified as a symptom of anxiety or depression (Flaskerud, 2010). With the rise of obesity in the 1990s, clinicians began noticing bingeing behaviours in the absence of compensatory purging behaviours or clinically relevant depression and anxiety (Devlin, Walsh, Spitzer, Hasin, 1992; Spitzer, 1991). It was, however, only when these behaviours mismatched the criteria of any other eating disorder that BED gained clinical validity (Flaskerud, 2010). As Davis (2013c) reviewed, BED was originally listed in the DSM-IV (American Psychiatric Association, 1994) under the *Eating Disorder Not Otherwise Specified* category, and was then moved to the Appendix of the DSM-IV-TR (American Psychiatric Association, 2000) for "further study". Currently, the DSM-5 recognizes BED as an independent eating disorder and mental illness, and characterizes it by

uncontrolled and recurring episodes of abnormally abundant food consumption with feelings of LOC, shame, and guilt (American Psychiatric Association, 2013). These recurring episodes happen at a minimum of once a week for a period of three or more months (American Psychiatric Association, 2013). Although BED is linked with obesity and can be a predictor of weight gain (Avena, Bocarsly, & Hoebel, 2012), BED may also be present in the absence of obesity, and vice versa (Mitchell, 2015). BED is the most prevalent eating disorder worldwide (Mitchell, 2015), with an estimated lifetime prevalence of 1.7% (Kessler et al., 2014). While the exact cause of onset of BED is unknown, negative moods (i.e. anger, sadness, loneliness, disgust, shame, stress) have frequently been reported as triggers for the disorder (Nicholls, Devonport, & Blake, 2016; Stice, Akutagawa, Gaggar, & Ageas, 2000; Zeeck et al., 2011). BED also has a strong association with other DSM-5 disorders such as anxiety, ADHD, and substance use disorders (Davis & Carter, 2009). There is also evidence that BED patients are hyper-sensitive to reward and show elevated levels of DA signaling, as well as higher levels of impulsivity (Schag et al., 2011; Manwaring et al., 2011), and compulsively overeat (Davis et al., 2009; Davis et al., 2012; Wang et al., 2011)

As Davis (2013, 2017) has argued, some reviewers and critics of research in the FA field have provided flawed perspectives on existing evidence by conflating FA with BED, overeating, and/or obesity. This is not surprising given that a key characteristic of both FA and BED is a LOC over the intake of hyper-palatable foods (Davis et al., 2009, Davis et al., 2012; Gearhardt et al., 2011b; Schulte et al., 2016a; Wang et al., 2011), and that both disorders may subsume other psychopathological entities such as substance addiction, ADHD, depression, and anxiety (Brownley et al., 2016; Davis et al., 2013; Wang et al., 2011). Both BED and FA groups also display similar genetic profiles, wherein both YFAS-diagnosed food addicts (Davis et al., 2013b;

Gearhardt et al., 2011b; Pedram et al., 2017) and BED patients (Davis et al., 2012; Wang et al., 2011) show a genetic predisposition to increased DA neurotransmission, and enhanced food-related reward response.

Human research has demonstrated that while co-morbidity does exist between BED and FA, only about 50 (Davis et al., 2011) to 60 percent (Ivezaj, White, & Grilo, 2016) of BED patients also meet the diagnostic criteria for YFAS-FA, indicating that binge-eating behaviours are neither sufficient nor necessary for a FA diagnosis (Davis, 2017; Davis et al., 2011; Gearhardt et al., 2012; Ivezaj et al., 2016). One viewpoint is that among those with BED, FA may reflect a more "severe" form of the disorder. In other words, amongst certain vulnerable individuals, the development of binge-eating behaviours may ultimately lead to a more chronic condition that has strong clinical and biological parallels to clinically-established addiction disorders (Davis, 2013a). Preliminary support comes from a study showing that among a sample of BED patients, those with YFAS-FA had more severe and frequent binges and cravings, showed elevated levels of hedonic and emotional eating, impulsivity and addictive traits, and were more likely to have severe depression, negative affect, eating disorder psychopathology and lower self-esteem compared to their non-FA counterparts (Davis, 2013b; Gearhardt et al., 2012). Accordingly, Davis's (2013a) dimensional view of overeating proposes that food consumption patterns range from non-psychopathological homeostatic eating (energy-balanced eating ascribed mainly by a 'normal' BMI score) at the lowest end of the continuum to FA at the highest. Binge-eating behaviours start mid-way along this continuum (Davis, 2013c), and are characterized by the DSM-5 as either a feeling of LOC or excessive food consumption in a relatively short period of time (American Psychiatric Association, 2013). So, while binge eating can progress into an addictive behaviour, it is not as severe nor the only factor in the development of FA.

1.2.5.1 Sex-based Variations among Food Addiction and Binge Eating

Some human studies have focused on the role of sex differences in the development of both FA and BED, yet research remains understudied – especially in the field of FA – and mainly focuses on disparities in prevalence. Instead, it is the sex-based variations of patterns underlying both behaviours that have been more vigorously studied and will be comparatively discussed below. Importantly, while it can be said that sex plays a significant role in the etiology of overeating behaviours, some findings have been contradictory (Burton, Smit, & Lightowler, 2007; Forrester-Knauss & Stutz, 2012; see Guerdjikova, Mori, Casuto, & McElroy, 2017; Meule, Hofmann, & Weghuber, 2016).

Obesity, a predictor of both FA and BED (Avena, Rada, & Hoebel, 2012; Pursey et al., 2016; VanderBroek-Stice, 2017), is higher in women worldwide, particularly in the severe classes of obesity (World Health Organization, 2017). Since the overconsumption of hyperpalatable foods clearly increases the risk for obesity, and since the chronic intake of said foods can lead to binge-like or addictive consumption (Pursey, Davis, & Burrows, 2017; Schulte et al., 2015a), it is not surprising that a relationship between FA, BED and obesity exists. Accordingly, as obesity is higher in women, the prevalence of both BED (see Klump et al., 2017) and FA (Mies et al., 2017; see Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014) is significantly higher in women.

Research has suggested that sex hormones may be important modulators in the expression of overeating behaviours (Asarian & Geary, 2013; Brown, Gent, Davis, & Clegg, 2010; Hallam et al., 2012). Estrogen has been implicated in attenuated appetite and eating in both males and females (Brown et al., 2010), and cyclic decreases in estrogen levels – seen in females due to the menses – have been associated with food cravings and food-cue reactivity (Frank et

al., 2010; Hormes & Timko, 2011). Accordingly, the luteal phase of the menstrual cycle – which exhibits lower estrogen levels – has been consistently associated with increased levels of food cravings towards palatable foods, particularly to sweet foods (Asarian & Geary, 2013; Frank et al., 2010; Hormes & Tinko., 2011; Klump et al., 2014). As such, women show elevated neural activity to palatable food cues during postovulatory phases, in neural areas including the orbitofrontal cortex and insula (Frank et al., 2010). Similarly, women respond significantly more strongly than men to hedonic food cues in the lateral and dorsolateral prefrontal cortex (Cornier, Salzberg, Endly, Bessesen, & Tregellas, 2010). In accordance, researchers have found that food cravings are higher in women (Chao, Grilo, & Sinha, 2016; Imperatori et al., 2013). Women are also more likely to report feelings of depression, negative affect, anger, and solitude during episodes of food cravings (Moore & Zhang, 2011). With relation to BED and FA, research has shown that food cravings are higher in females (Davis et al., 2011; Davis et al., 2012a; Hallam et al., 2016; Imperatori et al., 2013).

That the pathogenesis of BED and FA is modulated by impulsivity has also been established (Koball et al., 2016; Pivaranus & Conner, 2015; VanderBroek-Stice, 2017). Research consistently suggests that the trait of impulsivity is higher in males (see Cross et al., 2011), yet, as previously mentioned, both overeating behaviours are more frequent in women. This may possibly be due to the fact that the impulsivity exhibited by males creates a motivation to engage in risk-taking behaviour. For example, males are more likely to gamble and engage in dangerous driving and illicit substance use (Cross et al., 2011), while women, who score higher in risk aversion and are more sensitive to the negative outcomes of risky behaviour (Cross et al., 2011), may manifest their impulsivity through the overconsumption of hyperpalatable foods.

1.2.6 A 'Grazing' Pattern of Overeating

As with other addictive tendencies, overconsumption can manifest as patterns of intake that do not involve episodes of bingeing but still have the characteristics of an addiction. Overeating in the form of *grazing* can represent such an eating pattern, characterized by the inability to resist repetitive snacking throughout the day (Carter & Jansen, 2012; Conceição et al., 2014; Saunders, 2004). Conceição et al. (2014) explain that the term was originally recognized as an eating pattern with possible diabetic complications (e.g. high blood sugar; Calles-Escandon, Jaspan, & Robbins, 1989), rather than as an eating behaviour that resulted in failed dieting attempts (Wittig & Wittig, 1993); poorer binge-eating treatment (Harvey, Rawson, Alexander, & Bachar, 1994); and unsuccessful gastric-bypass surgery outcomes (Cook & Edwards, 1999). With the recent rising interest in grazing (Carter & Jansen, 2012; Conceição et al., 2014; Davis, 2016a; Marek, Ben-Porath, & Heinberg, 2016; Nicolau, 2015; Parker & Brennan, 2015; Walsh, Attja, Glasofer, & Sysko, 2016), the term is now being defined as the "mindless" consumption of smaller food servings over an extended period of time (Carter & Jansen, 2012; Conceição et al., 2014; Saunders, 2004). Conceição et al. (2014) have provided an expanded definition by adding that this repetitive behaviour tends to be unresponsive to hunger/satiety signals. They have also defined 'repetitive' as occurring at least twice within the same day with no prolonged gaps (e.g. less than one hour) present between episodes. In addition, Conceição et al. (2014; 2017) have proposed two subtypes of grazing: (a) *compulsive grazing*, defined as the inability to stop repetitively snacking despite having the intention to do so, and (b) *non-compulsive grazing,* defined as repetitive bouts of mindless eating. While compulsive grazing is associated with pronounced cravings for tempting foods and is, to some degree, associated with LOC, the non-compulsive subtype is associated with eating any foods that are

available without a LOC (Conceição et al., 2014; Conceição et al., 2014). In an effort to shed theoretical clarity on these two subtypes, Goodpaster et al. (2016) found, in a sample of bariatric surgery candidates, that compulsive grazing versus non-compulsive grazing occurred an average of four days per week versus two days per week, and was associated with greater psychopathology, suggesting that grazing with LOC (compulsive grazing) is the more severe subtype. In a qualitative study, Zunker, Karr, Saunders and Mitchell (2012) alternatively found that the non-compulsive subtype of grazing is viewed as a positive adaptive behaviour in post-bariatric surgery patients.

Grazing has garnered the most interest in the context of outcomes following bariatric surgery (Conceição et al., 2014), where it is viewed as a "high-risk behaviour" (Saunders, 2004). It is important to note that grazing episodes are different than when post-operative bariatric patients intentionally eat multiple smaller meals as prescribed (Conceição et al., 2014; Conceição, Utzinger & Pisetsky, 2015). In the latter situation, both the choice of food and the time of ingestion are somewhat planned, controlled, and occur in response to hunger/satiety signals (Conceição et al., 2014; Conceição et al., 2015). With grazing, evidence suggests that increased rates often occur following surgery (Conceição et al., 2014), wherein the majority (Saunders, 1999; Saunders, 2004; Nicolau, 2015) of bariatric patients experience post-operative grazing episodes due to their diminished gastric capacity, and the concurrent inability to hold an overabundant mass of food (Saunders, 2014). Other studies have reported that grazing was related to significantly less weight loss and even weight regain following surgery (Colles, Dixon, & O'Brien, 2007; Kofman, Lent & Swencionis, 2010). Robinson et al. (2014) also found that grazing diminished treatment success in both patients who had low and moderate-to-high dietary adherence.

Grazing has also been associated with BED (Busetto et al., 2005; Goodpaster et al., 2015), binge eating (Colles et al., 2008; Saunders, 2004), LOC eating (Goodpaster et al., 2015; Conceição et al., 2014; Lane & Szabó, 2013; Saunders, 2004) and depression (Kofman et al., 2010; Nicolau et al., 2015). The relationship between grazing and binge eating suggests that gastric bypass patients who binge pre-operatively convert to grazing behaviours post-operatively (Colles et al., 2008; Saunders, 2004). While both binge eaters and grazers experience LOC, authors have suggested that the LOC associated with grazing is clinically different than the LOC experienced during a binge (Conceição et al., 2015). Similar constructs to grazing currently exist, such as picking and nibbling, however little research exists on the overlap between these conditions (Walsh, 2016). To date, the relationship between FA and grazing is unknown.

CHAPTER 2: THE CURRENT STUDY

Regrettably, grazing in the context of FA has not yet been studied. Moreover, additional research is needed to understand the associations among binge eating, LOC eating, reward-driven eating and FA, especially since some of these behaviours can co-exist and show strong overlap, and because the question of whether FA is a legitimate food-use disorder still exists. The current cross-sectional study was designed to explore the unique variance accounted for in the diagnosis and symptom count of FA by separate patterns of overeating including grazing, binge eating, reward-driven eating and LOC eating, after accounting for established physiological and psychological covariates.

Given the unexplained relationships in previous research, the current study model is exploratory in nature. Thus, overall predictions were made without prior empirical evidence. However, due to the clinical similarities between grazing, binge eating, and LOC eating (Colles

et al., 2008; Goodpaster et al., 2016), and the similarities between FA, binge eating, and LOC eating (Davis, 2013c; Gearhardt et al., 2011a), it was expected that grazing would make a unique contribution to the variance in FA, after accounting for binge eating, LOC eating, and reward-driven eating. In other words, it was predicted that these overeating behaviours would positively predict FA symptomology and diagnosis.

CHAPTER 3: STUDY DESIGN AND METHODOLOGY

3.1 Participants

Male and female adults ranging in age from 20 to 50 years were invited to participate. Students at York University (YU) were recruited through an internal online portal. Participation was part of a course requirement, where students received a credit upon their completion that contributed to their final course mark. Participants were recruited from two YU courses within two different departments in the Faculty of Health, each using a unique internal departmental online portal: (a) students from the "KINE 2049: Research Methods in Kinesiology" course in the School of Kinesiology and Health Science were recruited via the "Kinesiology Undergrad Research Experience" (KURE); and (b) students from the "PSYC 1010: Introduction to Psychology" course in the Department of Psychology were recruited via the "Undergraduate Research Participant Pool" (URPP). At the time of their in-lab appointment, participants must have been residing in North America for a minimum of five years, been fluent in English, and have had the ability to provide informed written consent. Participants were excluded from the study if they: (a) were pregnant or lactating, and/or (b) had physical disabilities. In order to increase sample size, a dataset from Griffith University (GU) in South East Queensland Australia was also used in this study. Participants from the GU sample were part of an independent

collaborative study conducted at GU following the aims and procedures of the YU study. Ethics approval for that study was administered at GU. Participants from the GU dataset were recruited via a study web link posted on social media sites (i.e. Facebook, Twitter) by GU and YU researchers, and an internal email sent to GU staff and students.

A total of 233 participants (nYU=152, nGU= 81) were originally recruited for the study. Participant data were excluded if sex, gender, or age were missing, or did not have the YFAS complete, giving a final total of 201 participants (nYU= 152, nGU= 49). Participants self-identified as 43% Caucasian (n=87), 18% African Descent (n=36), 17% East Indian Caucasian (n=35), 9% Oriental (n=18), and 10.9% as other (n=22; First Nations, North American Hispanic/Latino, Middle Eastern). Participants were 69% female (n=139) and 31% male (n=62). Mean age was 25 years, and mean BMI was 25.0 kg/m^2, ranging from 16.7 kg/m^2 to 41.4 kg/m^2. YFAS-FA was prevalent in 13% of participants.

3.2 Eating-Behaviour Measures

3.2.1 Food Addiction was assessed by the *Yale Food Addiction Scale* (YFAS), a 25-item self-report scale reflecting the 7 symptoms (viz. excessive time spent seeking and using the substance; taking a substance for longer than intended; persistently, unsuccessfully stopping substance usage; continued use despite knowledge of aversive consequences; continued use despite negative personal and interpersonal problems; tolerance; and withdrawal) of a substance-dependence disorder according to the DSM-IV (American Psychiatric Association, 2000) modified for eating behaviours (Gearhardt et al., 2009). The YFAS uses both dichotomous and continuous scoring options. Parallel to the DSM-IV, a ‘diagnosis’ of FA is indicated by the endorsement of 3 or more out of the 7 symptoms and by the “clinically significant impairment” criterion being met in the past 12 months. The continuous scoring option provides a symptom-

count score, and is given by summing the number of symptoms endorsed, meaning scores can range from 0 to 7 symptoms endorsed of the food-specific DSM-IV criteria. Example of items include, “I eat to the point where I feel physically ill” and “My behaviour with respect to food and eating causes significant distress”. Cronbach’s alpha for the YFAS symptom score in the current study was 0.84.

3.2.2 Binge Eating was assessed by the *Binge Eating Questionnaire* (BEQ), which measures the extent and severity of bingeing and purging behaviours (Halmi, Falk, & Schwartz, 1981). Since the current study only focuses on bingeing, rather than purging behaviours, only 5-items of the BEQ, which comprise the binge-eating subscale were used (see Halmi et al., 1981). Questions in this subscale identify binge-related symptoms such as “Do you ever get uncontrollable urges to eat and eat until you feel physically ill?”, and “Are there times when you are afraid that you cannot voluntarily stop eating?”. Binary scoring (yes-no) is used for each item. A higher score reflects more severe symptoms. Cronbach’s alpha for the BEQ in the current study was 0.75.

3.2.3 Loss of Control Eating was assessed by the 24-item *Loss of Control over Eating Scale* (LOCES), which measures LOC eating within the past month (Latner, Mond, Kelly, Haynes & Hay, 2014). Items are scored on a 5-point Likert scale ranging from 1 (never) to 5 (always). Questions cover 3 aspects of LOC eating: 1) behavioural manifestations (i.e. inability to stop eating and/or eating despite the presence of adverse consequences), 2) cognitions/dissociative features of LOC eating (i.e. sole focus on eating and reality distortion), 3) positive/euphoric features of LOC eating (i.e. feeling high or feeling relief). Example of items include “While eating, I felt disgusted” and “I felt like the craving to eat overpowered me”. Items

are averaged to obtain a total score, with a higher score indicating greater LOC eating. Cronbach's alpha of the LOCES in the current study was 0.96.

3.2.4 Grazing was assessed by the *Rep(eat) Questionnaire* (Rep-Q), which is a 12-item self-report measure used to operationalize the behaviours and cognitions of 'grazing' (Conceição et al., 2017). The scale was developed by 18 experts in the field of bariatric surgery and/or eating behaviours, and has been validated both in a non-clinical population and in a pre- and post-bariatric clinical population. Conceição et al. (2017) differentiate two sub-types of grazing: 1) non-compulsive and 2) compulsive. The non-compulsive subtype displays mindless, distracted eating with no LOC, and can, for example, include eating both a cookie and fruit as a snack. Items of the questionnaire that comprise the non-compulsive sub-type include 1, 2, 3, 4, 9 and 10, and include items such as "Snacked on food repetitively throughout the day" and "Ate without paying attention to the amount of food you were eating". The compulsive subtype reflects an inability to resist snacking and food cravings, and can, for example, include finishing an entire box of cookies. Items 5, 6, 7, 8, 11, and 12 of the questionnaire comprise the compulsive sub-type, and include items such as "Did not want to eat, but felt that you couldn't avoid eating" and "Had a hard time controlling your cravings to eat in between meals". Items are scored on a 6-point Likert-type scale with responses ranging from 0 (never) to 6 (usually every day). Total scores are the average of all individual item scores. A higher total average reflects more frequent grazing. With regard to the subscales, a higher average reflects more frequent compulsive or non-compulsive grazing. Cronbach's alpha of the Rep-Q in the current study was 0.93.

3.2.5 Reward-Based Eating was assessed by the 9-item *Reward-Based Eating Drive Scale* (RED) which measures reward-based eating – a non-pathological behaviour that is associated with LOC, preoccupation with food, and lack of satiation, and is rooted from neural circuitry which drives hedonic eating (Epel et al., 2014). The scale shows positive correlation with BMI, life-long obesity, and weight fluctuation, and has thus been recommended for use in identifying those at risk of overweight or obesity, and those at risk of yo-yo dieting. Items are distributed on a 5-point Likert scale ranging from 0 (very false) to 4 (very true) and are averaged to comprise a total score. Items capture 3 reward-related eating constructs: (1) lack of control over eating, (2) lack of satiation, and (3) preoccupation with food. Examples of items include "I feel out of control in the presence of delicious food" and "food is always on my mind". Items are averaged, and a higher score is positively correlated with greater reward based eating. Cronbach's alpha of the RED in the current study was 0.87.

3.3 Personality Measures

3.3.1 Impulsivity was measured by the 30-item *Barratt Impulsiveness Scale* version 11 (BIS-11; Patton, Stanford & Barratt, 1995), which measures impulsiveness on a 4-point scale ranging from 1 (rarely/never) to 4 (almost always/always). Currently, this is the most frequently used scale for self-identified impulsivity (Davis et al., 2011). The scale conceptualizes impulsivity according to 3 overarching dimensions: (1) attentional impulsiveness (e.g. "I get bored really easily"), (2) non-planning impulsiveness (e.g. I buy things on impulse"), and (3) motor impulsiveness (e.g. "I don't plan for the future"). Items are summed to obtain the total score. To avoid response bias, some items are scored in reverse order (e.g. "I plan for the future"). A higher score reflects more severe symptoms. Cronbach's alpha of the BIS-11 in the current study was 0.82.

3.3.2 Addictive Personality Traits was assessed by the *Addiction Scale* of the *Eysenck Personality Questionnaire-Revised* (EPQ-R; Eysenck & Eysenck, 1991). This scale was derived by identifying items on the EPQ-R that differentiated drug addicts from controls at a probability level less than or equal to 0.001 (Gossop & Esyenck, 1980). The Addiction Scale contains items from four EPQ-R subscales: 4 items from the 'Extraversion-Introversion' scale (e.g. "Do you enjoy meeting new people?"), 13 items from the 'Neuroticism' scale ("Have you ever wished that you were dead?"), 9 items from the 'Psychoticism' scale (e.g. "Have you ever taken advantage of someone?"), and 6 items from the 'Lie' scale (e.g. "Would you dodge paying taxes if you were sure you could never be found?"). Answers are scored using a dichotomous yes-no format. To avoid response bias, 8 items are scored in reverse direction. A higher score reflects more addictive personality traits. Cronbach's alpha of the Addiction Scale in the current study was 0.79.

3.4. Physical Measures

3.4.1 Height and Weight were measured with the participant wearing light clothing and standing in stocking feet on a physician scale. BMI (kg/m^2) was calculated by dividing the measured weight (kg) by the square of measured height (m^2).

3.5 Procedures

YU participants responded to a posting listed on an internal course website (KURE or URPP), where completion granted them a credit that counted towards their final mark. The posting instructed participants to initially identify if they had met pre-requisite criteria, and then to choose from a list of available time-slots to come into the research coordinator's laboratory to participate in the study. Available time-slots were pre-set by the research coordinator. Upon arrival at the laboratory, participants were first required to read and sign an informed consent

document (see Appendix A), and were then asked a list of questions in a face-to-face interview to confirm their prerequisite criteria and demographic characteristics (see Appendix B). Participation in the study then required completing the self-report questionnaire booklet, which comprised the eating behaviour and personality measures listed above, in a private room in the laboratory. After completing the questionnaire booklet, height and weight were measured and recorded. Participants were given as much time as they needed to complete the booklet, and were allowed to ask the researcher any questions they had. To avoid response bias, questionnaire order was randomized across participants.

Participants in the GU study responded to an online web link posted by GU and YU researchers to various social media websites (e.g. Facebook, Twitter). The posted web link included a short accompanying paragraph describing the study. In order to increase recruitment, the research coordinator of the current study also posted the web link to targeted Facebook 'groups' such as "Overeaters Anonymous", "Struggling with Food Addiction", and "Beating Food Addiction For Good". GU researchers also sent out the web link in an email to staff and students at GU. GU researchers used an internal web survey tool called "Griffith University Research Survey Tool". Participants were required to click on the web link and fill in the questionnaire online. The introduction page of the study link included an information-and-consent document. Partaking in the study meant that the document was read and consent was given. No compensation was given for GU participants. The GU dataset was shared with researchers of the current study through an SPSS and Microsoft Excel file sent via email.

3.6 Statistical Analyses

All analyses for this study were done using *Statistical Package for the Social Sciences (SPSS) Version 23* for Windows (SPSS Inc., Chicago, IL, USA) at a two-tailed significance level of $\alpha = 0.05$. The GU dataset was received via email in an SPSS file. List-wise deletion was used in the GU dataset when sex, gender, and/or age were missing, and/or if the YFAS questionnaire was incomplete. Missing data in the YU dataset were imputed using information from related observations in corresponding scales or subscales. Data from the GU dataset were then merged with the YU SPSS file using the 'Merge Files' option in SPSS. Since data were taken from two separate samples (YU and GU), independent samples t-test analyses were performed on relevant demographic characteristics and scale total scores in order to assess group differences. YU participants were significantly younger (M = 23.15, SD = 4.88) than GU participants (M = 29.37, SD = 8.35), $t(199) = -4.95$, $p < 0.0001$. No other differences were found, thus, the datasets were integrated as one.

Independent samples t-test analyses of the entire sample were performed to examine sex differences in the predictor model variables (BMI, addictive personality traits, impulsivity, LOC eating, reward-driven eating, binge eating, and grazing) and the continuous YFAS variable. Accordingly, a chi squared (χ^2) test was performed to examine sex differences in the binary YFAS variable. Multiple regression analysis was used examine the predictive power of LOC eating, reward-driven eating, binge eating, and grazing on the continuous symptom-count variable of the YFAS. Logistic regression analysis was employed to estimate the associations of BMI, addictive personality traits, impulsivity, LOC eating, reward-driven eating, binge eating, and grazing on the odds of being diagnosed with YFAS-FA.

CHAPTER 4: RESULTS

4.1 Descriptive Statistics

Table 1 presents means and standard deviations, and the bivariate correlation coefficients among the model variables for the total sample. Normality was assessed by examining skew and kurtosis values, and by visual inspection of the distributions via histograms. All variables in the model were approximately normally distributed according to acceptable limits of ±2 for skew and kurtosis (Field, 2009; Gravetter & Wallnau, 2014).

Table 1: *Means, standard deviations, and correlation coefficients for the model variables*.

Variable	N	$\bar{x}$	SD	1	2	3	4	5	6	7	8
1. AS	195	13.32	10.27	–	0.47**	0.47**	0.101	0.50**	0.50**	0.51**	0.53**
2. BEQ	201	1.71	1.55	0.47**	–	0.43**	0.23**	0.77**	0.69**	0.64**	0.58**
3. BIS-11	201	62.46	10.26	0.47**	0.43**	–	0.11	0.54**	0.51**	0.49**	0.43**
4. BMI	201	25.0	4.2	0.10	0.23**	0.11	–	0.30**	0.24**	0.28**	0.24**
5. LOCES	198	4.63	1.86	0.50**	0.77**	0.54	0.30**	–	0.78**	0.80**	0.74**
6. RED	195	1.46	0.97	0.50**	0.69**	0.51**	0.23**	0.78**	–	0.75**	0.68**
7. REPQ	195	1.73	1.31	0.51**	0.64**	0.49**	0.28**	0.80**	0.75**	–	0.72**
8. YFAS[1]	195	2.54	1.82	0.53**	0.58**	0.43**	0.24**	0.74**	0.68**	0.72**	–

AS: Addiction Scale; BEQ: Binge Eating Questionnaire; BIS-11: Barratt Impulsiveness Scale-11; BMI: body mass index; LOCES: Loss of Control Over Eating Scale; RED: Reward-Based Eating Drive Scale; REPQ: Repetitive Eating Questionnaire; YFAS: Yale Food Addiction Scale.
** = $p < 0.01$.
[1]Symptom scores reported.

4.2 Independent Samples T-Test Analyses and Chi-Squared Test

Table 2 presents the means, standard deviations, minima, maxima, and independent samples t-test results for the quantitative variables used in the model, listed separately by sex. Independent samples t-test analyses were used to assess differences between males and females. Heteroscedasticity was present in the grazing (REPQ) variable, therefore, the *p*-value assuming unequal variance was reported. Results indicated a significant effect for BMI, with males having

higher values than females. For grazing, females had higher mean scores than males. No other main effects were found. A chi-squared test was conducted between sex and FA diagnosis and null results were obtained (χ^2 (1) = 5.68, p = 0.577).

Table 2: *Means, standard deviations, minima, maxima, and independent samples t-test analyses for quantitative variables, divided by sex.*

	Female					Male						
Variable	N	$\bar{x}$	SD	Min	Max	N	$\bar{x}$	SD	Min	Max	*t*	*p*
Age	139	26	7.0	20	50	62	24	5.2	20	50	1.09	0.279
AS	133	13.80	5.48	2.00	27.00	62	13.26	5.72	3.00	23.00	1.91	0.065
BEQ	139	1.81	1.59	0.00	5.00	62	1.82	1.62	0.00	5.00	1.40	0.164
BIS-11	139	62.74	10.65	40.00	96.00	62	64.39	10.54	40.00	88.00	0.58	0.566
BMI	139	24.4	4.0	16.8	36.6	62	26.5	4.6	16.7	41.4	-3.29	0.001
LOCES	136	1.90	0.81	1.00	4.63	62	1.92	0.88	1.00	4.00	1.19	0.234
RED	133	1.53	1.01	0.00	4.00	62	1.58	1.06	0.00	3.00	1.48	0.142
REPQ	133	1.88	1.40	0.00	5.92	62	1.68	1.55	0.00	4.50	2.53	0.012
YFAS[1]	139	2.63	1.89	0.00	7.00	62	2.32	1.83	0.00	7.00	0.92	0.361

AS: Addiction Scale; BEQ: Binge Eating Questionnaire; BIS-11: Barratt Impulsiveness Scale-11; BMI: body mass index; LOCES: Loss of Control Over Eating Scale; RED: Reward-Based Eating Drive Scale; REPQ: Repetitive Eating Questionnaire; YFAS: Yale Food Addiction Scale.
[1]Symptom scores reported.

4.3 Multiple Regression Analyses

The assumptions of multiple regression analysis include: (1) that a linear relationship exists between the outcome and predictor variable, (2) absence of influential cases, (3) independence of residuals, (4) homoscedasticity of residuals, (5) normality of residuals, and (6) absence of multicollinearity (Cohen, Cohen, West, & Aiken, 2002). Assumption 1 was tested by visual inspection of scatterplots, all variables passed the assumption. Assumption 2 of influential cases was tested using Cook's Distance values. All values were well under 1, suggesting that no individual cases were influencing the model, and that this assumption was also met. Assumption 3 was tested using the Durbin-Watson statistic and was met (Durbin-Watson = 2.00). To test the fourth assumption of homoscedasticity and fifth assumption of residual normality, a scatterplot

of the standardized predicted values against the standardized residuals, and a P-P plot was generated. Visual inspection of both plots suggested that these assumptions were met. Assumption 6 was tested by looking at the correlation matrix and the variance inflation factor (VIF) in the model (see Table 3). The correlation between LOC eating and grazing suggested the presence of multicollinearity, when following the suggested threshold value of 0.80 (Berry & Feldman, 1985). Due to this, the multiple regression analyses were run with and without the LOC eating variable in the model, as discussed below.

In order to examine the predictive power of LOC eating, reward-driven eating, binge eating, and grazing on FA, a multiple regression analysis was employed using the continuous symptom count variable of the YFAS as the dependent variable. Based on previous research (Davis et al., 2013; Gearhardt et al., 2011c; Long et al., 2015; Pedram et al., 2017; Pursey et al., 2016; VanderBroek-Stice, 2017), BMI, impulsivity, and addictive personality traits were included as covariates in the model and entered into Block 1, followed in Block 2 by measures of general overeating as reflected in the LOC eating and reward-driven eating variables. Finally, the binge eating and grazing variables were placed in Block 3. The model was also re-run using the subscales (compulsive and non-compulsive) of the grazing scale (REPQ) in Block 3. The adjusted R^2 for the final model was 0.61. Greater symptom severity was associated with higher addictive-personality traits, LOC eating, reward-driven eating, and grazing, with LOC eating making the largest contribution to the model, followed by grazing, addictive personality traits, and reward-driven eating, respectively. VIF values were also recorded to check for the presence of multicollinearity, especially in the LOC eating variable. While the VIF score of LOC eating was well below the most commonly suggested cut-off value of 10 (Cohen et al., 2002, see O'Brien, 2007), it has been suggested that in the behavioural sciences, this cut-off is too high and

should be interpreted with caution (see Chatterjee & Hadi, 2013; Cohen et al., 2002; O'Brien, 2007), so the model was re-run without the LOC variable. A summary of the results is presented in Table 3.

Similar patterns were observed when LOC eating was taken out of the model $F(6, 188) = 44.96$, $p < 0.0001$), with addictive personality traits ($\beta = 0.17$, $p = 0.004$), reward-driven eating ($\beta = 0.25$, $p = 0.002$), and grazing ($\beta = 0.41$, $p < 0.0001$) each making a statistically significant contribution in the final model. The adjusted R^2 for this model was 0.58.

Table 3: *Regression coefficients and VIF for the multiple regression analysis with YFAS symptom score as the dependent variable.*

Model 1

Variable	B	SE	*β*	*t*	*p*	VIF
AS	0.14	0.02	0.40	6.11	< 0.0001	1.28
BIS-11	0.04	0.01	0.22	3.36	0.001	1.28
BMI	0.08	0.03	0.18	3.13	0.002	1.02

Model 2

Variable	B	SE	*β*	*t*	*p*	VIF
AS	0.06	0.02	0.19	3.32	0.001	1.48
BIS-11	-0.01	0.01	-0.04	-0.63	0.528	1.55
BMI	0.01	0.02	0.03	0.59	0.560	1.11
LOCES	1.12	0.18	0.49	6.16	< 0.0001	2.94
RED	0.42	0.14	0.22	2.93	0.004	2.69

Model 3

Variable	B	SE	*β*	*t*	*p*	VIF
AS	0.06	0.02	0.17	3.01	0.003	1.53
BIS-11	-0.01	0.01	-0.05	-0.81	0.419	1.56
BMI	0.01	0.02	0.17	0.35	0.725	1.12
LOCES	0.91	0.22	0.40	4.07	< 0.0001	4.69
RED	0.31	0.15	0.17	2.09	0.038	3.08
BEQ	-0.09	0.09	-0.08	-1.04	0.300	2.60
REPQ[1]	0.36	0.11	0.26	3.22	0.002	3.26

AS: Addiction Scale; BEQ: Binge Eating Questionnaire; BIS-11: Barratt Impulsiveness Scale-11; BMI: body mass index; LOCES: Loss of Control Over Eating Scale; RED: Reward-Based Eating Drive Scale; REPQ: Repetitive Eating Questionnaire; YFAS: Yale Food Addiction Scale.
B: unstandardized coefficient; SE: standard error; *β*: standardized coefficient; VIF: variance inflation factor.
Model 1 Adjusted $R^2 = 0.34$.
Model 2 Adjusted $R^2 = 0.59$.
Model 3 Adjusted $R^2 = 0.61$.
[1]Only the compulsive subscale of the REPQ added significance in the model ($\beta = 0.41$, $p < 0.0001$). Results not shown in table.

4.4 Logistic Regression Analyses

In order to further examine the FA construct, logistic regression was employed to assess the probability of LOC eating, reward-driven eating, binge eating, and grazing in predicting the diagnosis of the logit transformation of the YFAS-FA binary variable (1 = yes, 0 = no). Similar to the multiple regression model, BMI, impulsivity, and addictive personality traits were included as covariates in the model and entered into Block 1, followed in Block 2 by measures of general overeating as reflected in the LOC eating and reward-driven eating variables. Finally, the binge eating and grazing variables were placed in Block 3. Since, as previously discussed, the LOC variable had a relatively-high VIF score, to be conservative, the logistic regression analysis was also re-run without the LOC variable in the model.

The logistic regression equivalent of R^2 – the pseudo R^2 – is an approximation of the coefficient of determination, and is based on the log likelihood of a model in comparison to the log likelihood of its baseline model (Hosmer et al., 2000; Long, 1997). In this analysis, the Nagelkerke R^2 was reported due to its ability to cover the range of zero to one, inclusive. Notably, the pseudo R^2 is not an equivalent statistic to the R^2 which follows an ordinary-least-squares approach to goodness of fit (Hosmer et al., 2000; Long, 1997). Rather, the pseudo R^2 is a maximum likelihood estimate, and is suggested to be interpreted as a measure of effect size of weak, moderate, or strong model fit, at cutoffs of 0.00 to 0.29, 0.30 to 0.59, and 0.60 to 1.0 respectively (Bewick, Cheek & Ball, 2005; Long, 1997).

Table 4 presents the logistic regression analysis when LOC eating was kept in the model. Results indicated that it was the only variable that significantly improved the fit of the logistic regression model compared to its baseline, providing a moderate fit in predicting FA diagnosis. Table 5 presents the logistic regression analysis when LOC eating was taken out of the model. In

this modified model, reward-driven eating was the only variable that reached statistical significance, with the overall model providing a weak fit in predicting FA diagnosis.

Table 4: *Regression coefficients and odds ratios for the logistic regression analysis with YFAS diagnosis as the dependent variable, and with LOCES kept in the model.*

Model 1

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Upper
AS	0.15	0.05	8.13	0.004	1.16	1.05	1.29
BIS-11	0.05	0.02	4.41	0.036	1.05	1.00	1.10
BMI	0.14	0.05	7.39	0.007	1.15	1.04	1.28

Model 2

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Lower
AS	0.09	0.06	2.36	0.124	1.10	0.98	1.24
BIS-11	-0.05	0.04	1.52	0.217	0.96	0.89	1.03
BMI	0.02	0.07	0.07	0.798	1.02	0.89	1.16
LOCES	1.82	0.56	10.49	0.001	6.19	2.05	18.63
RED	0.61	0.49	1.56	0.211	1.85	0.71	4.84

Model 3

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Lower
AS	0.10	0.06	2.46	0.117	1.10	0.98	1.24
BIS-11	-0.04	0.04	1.43	0.231	0.96	0.89	1.03
BMI	0.02	0.07	0.10	0.756	1.02	0.90	1.17
LOCES	1.91	0.68	7.78	0.005	6.72	1.76	25.64
RED	0.65	0.52	1.59	0.207	1.92	0.70	5.23
BEQ	-0.01	0.23	0.00	0.982	0.99	0.60	1.65
REPQ	-0.10	0.32	0.09	0.759	0.91	0.49	1.69

AS: Addiction Scale; BEQ: Binge Eating Questionnaire; BIS-11: Barratt Impulsiveness Scale-11; BMI: body mass index; LOCES: Loss of Control Over Eating Scale; RED: Reward-Based Eating Drive Scale; REPQ: Repetitive Eating Questionnaire; YFAS: Yale Food Addiction Scale.
SE: standard error; OR: odds ratio; CI: confidence interval.
Model 1 Nagelkerke $R^2 = 0.29$, $\chi^2 = 33.61$.
Model 2 Nagelkerke $R^2 = 0.53$, $\chi^2 = 65.53$.
Model 3 Nagelkerke $R^2 = 0.53$, $\chi^2 = 65.62$.

Table 5: *Regression coefficients and odds ratios for the logistic regression analysis with YFAS diagnosis as the dependent variable, and with LOCES omitted from the model.*

Model 1

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Upper
AS	0.15	0.05	8.13	0.004	1.16	1.05	1.29
BIS-11	0.05	0.02	4.41	0.036	1.05	1.00	1.10
BMI	0.14	0.05	7.39	0.007	1.15	1.04	1.28

Model 2

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Lower
AS	0.08	0.06	2.08	0.149	1.08	0.97	1.20
BIS-11	0.00	0.03	0.00	0.991	1.00	0.94	1.06
BMI	0.09	0.06	2.24	0.135	1.09	0.97	1.22
RED	1.53	2.30	13.10	< 0.0001	4.63	2.12	9.94

Model 3

						95% CI for OR	
Variable	Coefficient	SE	Wald	*p*	OR	Lower	Lower
AS	0.07	0.06	1.36	0.244	1.07	0.96	1.19
BIS-11	-0.02	0.03	0.24	0.627	0.98	0.92	1.05
BMI	0.05	0.06	0.74	0.390	1.06	0.93	1.19
RED	1.00	0.49	4.25	0.039	2.73	1.05	7.09
BEQ	0.32	0.23	1.92	0.166	1.38	0.88	2.18
REPQ	0.27	0.27	1.00	0.317	1.31	0.77	2.20

AS: Addiction Scale; BEQ: Binge Eating Questionnaire; BIS-11: Barratt Impulsiveness Scale-11; BMI: body mass index; LOCES: Loss of Control Over Eating Scale; RED: Reward-Based Eating Drive Scale; REPQ: Repetitive Eating Questionnaire; YFAS: Yale Food Addiction Scale.
SE: standard error; OR: odds ratio; CI: confidence interval.
Model 1 Nagelkerke $R^2 = 0.29$, $\chi^2 = 33.61$.
Model 2 Nagelkerke $R^2 = 0.44$, $\chi^2 = 53.70$.
Model 3 Nagelkerke $R^2 = 0.47$, $\chi^2 = 57.01$.

CHAPTER 5: DISCUSSION

The purpose of this cross-sectional study was to use multiple and logistic regression modeling to estimate the unique variance accounted for in the symptom count and the diagnosis of FA, respectively, by various overeating patterns and behaviours – most importantly binge eating and grazing – after taking account of well-established psychological and physiological covariates. While the study was largely exploratory in nature, it was known prior that associations among grazing, LOC eating, and binge eating (Colles et al., 2008; Conceição et al.,

2017; Goodpaster et al., 2016), and among FA, binge eating, and LOC have been demonstrated (Davis, 2013c; Gearhardt et al., 2011a). Therefore, it was also expected that grazing would make a unique contribution to the variance in FA. Although some research has found that FA is more prevalent in females (Burrows et al., 2017; Gearhardt et al., 2016; Pedram et al., 2013), this was not the case in the present study. Overall, FA was prevalent in 13% of participants.

5.1 Symptoms of Food Addiction

In the final multiple regression model (block 3), the addictive traits variable was the only significant covariate contributor to the variance in the symptom-count score of FA. The statistical significance of addictive personality traits reinforces the similarity of FA to other addiction disorders, which have also been strongly linked to the addiction personality scale (Mandic-Gajic, Dolic, Eror, & Spiric, 2017; Papachristou, Nederkoorn, & Jansen, 2016; Preller et al., 2013). Therefore, just as those who have high levels of addictive personality traits may be at a greater risk of engaging in traditional addictive behaviours, these same individuals may be more likely to establish a harmful relationship with food, especially in our obesogenic-food environment. As other work has also suggested, the links between FA and other addictive disorders, suggests that FA is better conceptualized as an addictive process than as an eating disorder (Davis, 2017; Gearhardt et al., 2011c; Hone-Blanchet & Fecteau, 2014).

Surprisingly, impulsivity did not make a significant contribution to the final model. While impulsivity on its own has been robustly associated with FA (Davis, 2010; Koball et al., 2016; Meule et al., 2017; Pivaranus & Conner, 2015) and an array of other drug and behavioural addictions (Sperry et al., 2016; Volkow & Fowler, 2000), it does have moderate shared variance with other personality measures, such as the addictive personality variable used in this study. That is, certain items reflecting impulsiveness are contained within the addictive personality

scale, which may be why impulsivity added no additional unique variance. More specifically, the addictive personality traits measure focuses most prominently on elements of "neuroticism" – such as emotional reactivity and negative affect – which have also been established as components of impulsivity (see Smith & Guller, 2014). Results of the current study also mesh well with Davis and Loxton's (2013), who found that while addictive personality traits were predictive of addictive behaviours overall, certain traits – such as negative affect and anxiousness – were stronger predictors than impulsivity. As previously discussed, the 'addictive personality' construct is multifaceted (Davis, 2016), and, may be a better predictor of FA than impulsiveness on its own.

While research has demonstrated that those with an FA diagnosis have a significantly higher BMI than their non-FA counterparts (Long et al., 2015; Pursey, Gearhardt, & Borrows, 2016; VanderBroek-Stice, 2017), it is perhaps not surprising that BMI was not a strong predictor of FA in the final model after accounting for other variables since the current sample largely comprised young, healthy-weight adults. It is important to emphasize that these results do not dispute that the habitual and compulsive intake of hyper palatable foods will, in most cases, lead to a greater BMI. Rather, the BMI of the current sample was lower than the global and Canadian adult population norm (World Health Organization, 2017; Parliament of Canada, 2016). It is also important to emphasize that the overconsumption of high-calorie foods is not equivalent to having addictive tendencies towards hyper-palatable foods, since the majority of the adult population falls in the overweight or obesity category, while only a small proportion of adults meet clinical diagnostic status for FA (World Health Organization, 2017; Pedram et al., 2013).

After being entered into the second model (block 2), LOC eating and reward-driven eating each made a statistically significant contribution to the variance in the YFAS symptom-

count variable. As outlined in the DSM-5, LOC – which refers to the increase in consumption of a substance relative to both quantity consumed and time spent consuming – is a hallmark of all addiction disorders (American Psychiatric Association, 2013). Previous studies have found that LOC contributes to the overconsumption of addictive substances (Volkow & Fowler, 2000; Volkow et al., 2010). Other authors have also suggested that deficits in inhibitory control may contribute to FA diagnoses (Hone-Blanchet & Fecteau, 2014) and an overlap between FA and LOC has been previously established (Epel et al., 2014; Lowe et al., 2016; Meule & Gearhardt, 2014). Results of the current study indicated the centrality of the LOC construct to the susceptibility of developing FA, while also underscoring the similarity between the behavioural symptoms exhibited in FA and other addictive behaviours.

Although this study was the first to assess the relationship between reward-driven eating and FA, previous FA studies have shown similar results (Davis, 2013b; Davis et al., 2013; Loxton & Tipman, 2017). For example, Davis (2013b) found that participants with FA were more likely to engage in hedonically-driven eating, while Davis et al. (2013) and Loxton and Tipman (2017) found a positive relationship between FA and elevated reward sensitivity. High reward sensitivity has also been associated with elevated DA signaling in other addictive behaviours (Dissabandara et al., 2012; Genovese & Wallace, 2007; Nestor et al., Garavan, 2010). Such findings suggest that those with FA tendencies exhibit a heightened susceptibility for reward sensitivity, and are more likely to engage in reward-driven eating-behaviours.

Finally, when grazing and binge eating were entered into the third and final model (block 3), only grazing made a significant contribution. These results are compelling since they are the first to show that individuals who self-report elevated addictive tendencies towards food also display elevated levels of grazing and snacking behaviours. Further inspection of the significance

of grazing demonstrated that it is only the compulsive, rather than the non-compulsive, component of grazing that significantly added to the variance in the present model. This is somewhat self-evident as compulsive overeating is a key aspect of FA (Gearhardt et al., 2011a; Joyner et al., 2015; Polk et al., 2017), and, since FA, as a concept of addictive tendencies towards food, is compulsive in nature. In line with the current study, Burrows et al. (2017) found that individuals with FA were more likely to report higher consumption of "snack foods" (e.g. chips, pastries, ice cream). Similarly, Davis et al. (2011) found that those with FA were more likely to snack on sweets. Taken together with the current findings, this may indicate that those with FA follow a certain dietary pattern, and that compulsive grazing may be a contributor in the spectrum of overeating leading to FA. Similarly, Conceição et al. (2017) suggest that compulsive grazing may be a central feature of disordered overeating. In addition, females also had significantly higher grazing scores, and, while sex-based disparities in prevalence were not found in FA in this sample, these results may still indicate that grazing and FA follow similar sex-based patterns, since there is good evidence from previous research that FA is more prevalent in females (Burrows et al., 2017; Gearhardt et al., 2016; Pedram et al., 2013). The significance of grazing in this study and its predominance over binge eating in predicting FA also emphasizes the similarity of FA to other addictive behaviors, where varied patterns of consumption – and not just bingeing – can lead to an addiction (Alonso-Alonso et al., 2015; Koob & Volkow, 2016).

While the bivariate relationship between binge eating and FA was significant in the current sample, binge eating did not make a significant contribution to the final model, after accounting for compulsive grazing. As with BMI, this may have happened due to a lack of binge-eating variance in this study's non-clinical sample, making binge eating scores lower than what has previously been found in clinical settings. For example, while the mean binge eating

score for the current sample was 1.71 (Table 1), using the same scale, the mean binge eating score in a sample of adults with obesity was 4.2 (Davis et al., 2011). Given the tendency of researchers to conflate BED and FA, it is important to emphasize that binge-eating does not necessarily warrant a FA diagnosis, that the two are not homogenous disorders, and that instead, other eating patterns and behaviours such as grazing, LOC eating, and reward-driven eating may be better predictors of FA.

5.2 Diagnosis of Food Addiction

In the logistic regression model, which was used to establish the predictive power of these variables in the *diagnosis* of FA, only LOC eating showed a significant association with the dependent variable, and only reward-driven eating significantly contributed to the model when LOC eating was removed. Psychometrically, this is not surprising given that the RED scale reflects LOC behaviours in its item content. The discrepancy in findings between the linear regression model and the logit model may simply be a reflection of lower power in the latter model. However, another possibility that might account for this difference is, that although the symptom count and diagnosis-scoring methods are clearly related, they measure different things, as further discussed below.

A Point-Biserial correlation in the current sample shows that the YFAS symptom-count score and the YFAS diagnosis are only correlated at a value of 0.58 ($p < 0.0001$). Some symptoms of the YFAS, such as "eating more than planned or for longer than planned", are likely to be endorsed by many in today's obesogenic-food environment, which is saturated with highly-palatable food (Armelagos, 2014; Finlayson & Dalton, 2012) and where portion sizes are bigger than ever (Steenhuis & Poelman, 2017). For example, this symptom was the most commonly reported and fluctuated most over time in a non-clinical longitudinal sample of adults

(Pursey et al., 2016). The symptom of a persistent desire to cut down on eating may also be endorsed by many individuals, especially since many people 'diet' (de Ridder, Adriaanse, Evers, Verhoeven, 2014). In Canada alone, the weight loss market is worth $189 million (Business Development Bank of Canada, 2013). As Rogers (2017) notes, having a difficulty in cutting down on certain foods seems more like normative behaviour rather than a symptom used to describe the pathological state of an addiction. Therefore, while the symptom-count score of the YFAS is useful in explaining the proneness and extent in which an individual is likely to engage in excessive and compulsive food-related behaviours, it does not quite measure the extremity of the addictive process and the distress and personal impairment with which that is associated.

In other words, to reach the diagnostic threshold of YFAS-FA, three or more symptoms must be endorsed, plus, the clinically significant impairment criterion as well. That is, regardless of how many symptoms are endorsed, a diagnosis cannot be made if a state of distress is not reported. Excessive food consumption may cause little to no impairment in many, while few may actually develop a condition that significantly impacts life quality. Acknowledging that FA can cause significant distress is especially pertinent in terms of evaluating whether it can be appropriately labelled as an addiction in the DSM. That is, the YFAS symptom-count score may simply serve as an indication of the degree to which an individual is likely to engage in the excessive consumption of hyper-palatable foods, while a YFAS diagnosis directly addresses whether this behaviour causes significant distress, and can appropriately be labelled as an addiction disorder. Therefore, and given that the two scoring methods are frequently used interchangeably, it is strongly suggested that they be treated as unique scoring methods establishing two different – yet related – phenomena.

5.3 Limitations and Future Directions

While this study is the first to establish a relationship between grazing and FA and between reward-driven eating and FA, these findings must be considered within the study's limitations. Firstly, the cross-sectional nature of this study makes it difficult to ascertain whether these eating behaviours and psychobehavioural characteristics were established as a consequence of FA, or whether they were present prior. Clinical recommendations are best made using longitudinal data due to their ability to elucidate these casual effects. Following individuals longitudinally is especially pertinent in the study of FA, where animal models have provided evidence that hyper-palatable foods have the ability to change neural reward-pathways and increase cravings (Avena & Hoebel, 2003; Carter et al., 2016). It is recommended that the current findings be replicated prospectively in order to establish that higher levels of addictive personality traits, LOC eating, reward-driven eating, and grazing can lead to higher FA symptomology, or that LOC eating and reward-driven eating can lead to a FA diagnosis. However, it is acknowledged that this approach is unlikely to be feasible on a large scale.

Participant responses may have also been underreported due to social desirability (e.g. highest weight, frequency of binges), especially in the online sample where weight was not confirmed by the researchers. However, our statistical analyses revealed no significant difference between self-report versus in-person measures of BMI. The online sample was partially obtained through targeted recruitment, since the study link was posted in various overeating-behaviour related Facebook groups. The undergraduate sample may also have been subject to targeted recruitment, as students had the option of signing up for studies they were interested in. It may be that those who chose to complete the study were more interested in food-related behaviours, which could have contributed to selection- and non-response bias. The study was also largely

skewed toward the lower end of the selected age-range and represented a relatively low BMI, probably because the majority of the sample was obtained from healthy-weight undergraduate students, and because most undergraduate students are not mature students. The homogeneity of the sample reduces this study's generalizability. It is therefore strongly encouraged that the study be replicated using a wider representation of individuals before any firm conclusions can be drawn.

Lastly, it is important to emphasize that a relatively small number of participants met the diagnostic criteria for FA, which reduces the power of the findings. However, the ability to find significance even when the sample of those meeting FA diagnostic criteria was small highlights the robustness of the observations, since the smaller the sample the lower the power to detect a substantial difference. Regrettably, the small sample of those meeting diagnostic FA status did not allow for assessing the possible moderating effects of sex. Such investigations are important, since worldwide increases in obesity – especially in the severe classes – are highest in women (World Health Organization, 2017). In addition, prevalence rates of both BED (see Klump et al., 2017) and FA (see Pursey et al., 2014; Mies et al., 2017) have been previously reported as being higher in women as well. Moreover, research from the addictions field indicates that the progression of a drug addiction is faster in women (Khan et al., 2013; Lewis, Hoffman, & Nixon, 2014), that women have poorer addiction-treatment outcomes (DeVito, Babuscio, Nich, Ball, & Caroll, 2014), and that women are subjectively more affected by their addiction (Griffin et al., 2015; Sherman et al., 2017). Animal models have also provided evidence that cyclic variations in estrogen levels mediates female drug seeking behaviour (Johnson & Kenny, 2010), and that female animals are more responsive to operant conditioning methods in relation to cocaine and amphetamine intake (Becker, Molenda, & Hummer, 2001). Understanding the mediating effects

of sex on FA is especially interesting in today's world, where – as anthropological and sociological literature attests – there are gender-related differences with regard to body image, social eating, and dietary choices (Araganini et al., 2012).

5.4 Summary and Conclusions

Despite these limitations, the current findings provide novel insight into the association between relevant eating patterns and behaviours, psychobehavioural characteristics, and FA. In this study, addictive personality traits, LOC eating, reward-driven eating, and grazing accounted for 61% of the variance in YFAS symptom count, and LOC eating accounted for 53% of the model fit for the diagnostic status of the YFAS, both of which are substantial findings. Taken together with Burrows et al.'s (2017) findings, it may be that those with FA are especially prone to a grazing-like dietary pattern, even in those who regularly binge eat. The current study is also the first to directly associate reward-driven eating with FA, although it should be noted that related associations have been previously reported (Davis, 2013b; Davis et al., 2013; Loxton & Tipman, 2017). While the binge-eating variable was not significant in any of the final models, this may have simply been due to insufficient variance in the current healthy-weight sample. Finally, due to the discrepancy in results between the logistic and multiple regression analyses, it is stressed that the YFAS symptom count score and YFAS diagnosis should not be used interchangeably as conceptually-equivalent constructs.

While future research is needed to assess the directionality of these relationships and confirm some preliminary findings of the current study, the current findings add to the increasingly compelling picture that the consumption of hyper-palatable foods can foster addictive-like eating behaviours in vulnerable individuals. More specifically, food-related behaviours appear to have the potential to be driven by compulsion, a LOC, and reward-related

mechanisms. With deliberations on an appropriate label for the so-called ‘food addiction’ construct still persisting, it is simply important to acknowledge that the consumption of hyper-palatable foods can become an addictive process.

In conclusion, it is relevant to re-consider our current obesogenic-food environment when discussing these addictive-like food-related behaviours. While some may feel autonomous in their eating decisions, it cannot be disputed that food consumption is habitual and driven by socioenvironmental cues (Gearhardt et al., 2011c; Johnson, 2013). In our society, food-related cues are omnipresent on televisions, the internet, social-media, and street signs; food advertisements have been shown to influence food-related decisions and consumptions (Burger & Stice, 2014; Yokum, Gearhardt, Harris, Brownell, & Stice, 2014). Therefore, it is important to acknowledge that for certain vulnerable individuals, making rational food choices can be especially challenging in this type of environment where hyper-palatable foods and their cues are so superfluous – especially if they are prone to poor inhibitory control and a hypersensitivity towards rewarding stimuli. Acknowledging and accepting this can have a large impact on public health, most importantly on the availability of treatments available to those with FA, and in the stigma surrounding obesity.

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Appendix A

INFORMED CONSENT FORM

DATE: _____

TITLE: Patterns of overeating that characterize addictive tendencies towards palatable foods

INVESTIGATORS:

1. Dr. Caroline Davis, Dept. of Kinesiology and Health Sciences, York University
2. Revi Bonder, MSc Candidate, Dept. of Kinesiology and Health Sciences, York University

PURPOSE: This study has been designed to determine whether a relationship exists between food addiction and other eating behaviours in adult men and women between 20 to 50 years old.

You are being asked to participate in this research study voluntarily. Before you agree to do this, it is important that you understand the study procedures, that you be aware of any benefits, risks, or discomforts that may be associated with your participation, and that you understand your right to refuse to participate or withdraw from this study. This is known as the informed consent process. This study has been reviewed and approved with the context of the Human Participants Review Subcommittee (HPRC) of York University and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines.

Please ask the researcher to explain anything that you don't understand before signing the consent form, and make sure all your questions have been answered satisfactorily before signing this document.

PROCEDURES: This study involves filling out a series of questionnaires and completing a physical assessment (weight and height measurement). You will be required to come into the lab once, and tasks will take approximately 45 minutes to complete.

RISKS AND DISCOMFORTS: We do not foresee any risks or discomforts from your participation in this study, however, if you experience any feelings of distress or psychological discomfort you may speak to a counselor at Personal Counseling services, N110, Bennett Centre for Student services, York University (telephone 416-736-5297) or at the York University Psychology Clinic (YUPC), Behavioural Sciences Building, Reception Room 104, York University (telephone 416-650-8488).

BENEFITS: There are no direct benefits in participating in this study other than having the knowledge of the relationship between food addiction and other overeating behaviours.

COMPENSATION: If you are a KINE 2049 Research Methods in Kinesiology student, and are a member of the Kinesiology Undergraduate Research Experience (KURE) participant pool, you will receive a 3% bonus mark for participating in this study.

WITHDRAWAL: You may stop participating in the study at any time, for any reason, if you so decide. Your decision to stop participating, or to refuse to answer particular questions, will not affect your relationship with the researchers, York University, or any other group associated with this project, either now or in the future. In the event that you withdraw from the study, all associated data collected will be immediately destroyed wherever possible. If you are a KINE 2049 Research Methods in Kinesiology student, and are a member of the Kinesiology Undergraduate Research Experience (KURE) participant pool, you will still receive a 3% bonus mark if you choose to withdraw from this study.

CONFEDENTIALITY: All information obtained during the study will be held in strict confidence. Your name or identifying information will not be used in any publication or presentation. Data will be stored for 10 years in a computerized encrypted SPSS file, and hard copies will be kept securely in the professor's locked laboratory. Only research staff will have access to the study's information. Data will be destroyed after 10 years through shredding and/or deletion from the databases. Confidentiality will be provided to the fullest extent possible by law.

QUESTIONS: If you have any questions about the study, please contact Dr. Caroline Davis either by phone or by email, or Revi Bonder by email. This research has received ethics review and approval by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. If you have any questions about this process, or about your rights as a participant in the study, please contact the Sr. Manager & Policy Advisor for the Office of Research Ethics, 5th Floor, Kaneff Tower, York University (telephone 416-736-5914 or e-mail ore@yorku.ca).

CONSENT: I _____ (please print full name) voluntarily consent to participate in Patterns of overeating that characterize addictive tendencies towards palatable foods conducted by Dr. Caroline Davis and Revi Bonder. I have had the opportunity to discuss this study and my questions have been answered to my satisfaction. I have understood the nature of this project and wish to participate. I consent to partake in this study with the understanding that I may withdraw at any time without prejudice. I am not waiving any of my legal rights by signing this form. My signature below indicates my consent. I have received a signed copy of this consent form.

_____	_____	_____
Participants name (please print)	Participants signature	Date

I confirm that I have explained the nature and the purpose of the study to the subject named above. I have answered all questions.

_____	_____	_____
Principal Investigator Name	Signature	Date

Appendix B

Demographic Information

GENDER: ☐ Male**:** ☐ Female

HEIGHT (Meters): _____

WEIGHT (Kilograms): _____

AGE: _____

RACE/ETHNICITY:
☐ European/Caucasian
☐ African Descent
☐ East Indian Caucasian (e.g. Pakistani, Indian)
☐ Oriental (e.g. Chinese, Japanese)
☐ North American Hispanic/Latino
☐ First Nations
☐ Other (please specify): _____

How many years have you lived in North America? _____

LEVEL OF EDUCATION:
☐ High school (completed)
☐ College/University (completed or some courses)
☐ Bachelor's degree (completed or in progress)
☐ Master's degree (completed or in progress)
☐ PhD., law, or medical degree (completed or in progress)
☐ Other (please specify): _____

ARE YOU CURRENTLY A REGULAR CIGARETTE SMOKER?

DO YOU REGULARLY DRINK ALCOHOL?

DO YOU REGULARLY USE RECREATIONAL DRUGS?